WEST Search History

DATE: Monday, October 07, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,DWPI; PLUR=YES; OP=ADJ		
L3	(L1 and (transgen$ or disrupt$ or knockout)) AnD ((@pd > 20020308)!)	10	L3
L2	(retina-specific nuclear receptor) AnD ((@pd > 20020308)!)	2	L2
L1	(retina-specific nuclear receptor or RNR) AnD ((@pd > 20020308)!)	39	L1

END OF SEARCH HISTORY

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NEWS 1       Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 Sep 17 IMSworld Pharmaceutical Company Directory name change
         to PHARMASEARCH
NEWS 3 Oct 09 Korean abstracts now included in Derwent World Patents
         Index
NEWS 4 Oct 09 Number of Derwent World Patents Index updates increased
NEWS 5 Oct 15 Calculated properties now in the REGISTRY/ZREGISTRY File
NEWS 6 Oct 22 Over 1 million reactions added to CASREACT
NEWS 7 Oct 22 DGENE GETSIM has been improved
NEWS 8 Oct 29 AAASD no longer available
NEWS 9 Nov 19 New Search Capabilities USPATFULL and USPAT2
NEWS 10 Nov 19 TOXCENTER(SM) - new toxicology file now available on STN
NEWS 11 Nov 29 COPPERLIT now available on STN
NEWS 12 Nov 29 DWPI revisions to NTIS and US Provisional Numbers
NEWS 13 Nov 30 Files VETU and VETB to have open access
NEWS 14 Dec 10 WPINDEX/WPIDS/WPIX New and Revised Manual Codes for
2002
NEWS 15 Dec 10 DGENE BLAST Homology Search
NEWS 16 Dec 17 WELDASEARCH now available on STN
NEWS 17 Dec 17 STANDARDS now available on STN
NEWS 18 Dec 17 New fields for DPCI
NEWS 19 Dec 19 CAS Roles modified
NEWS 20 Dec 19 1907-1946 data and page images added to CA and CAplus
NEWS 21 Jan 25 BLAST(R) searching in REGISTRY available in STN on the
Web
NEWS 22 Jan 25 Searching with the P indicator for Preparations
NEWS 23 Jan 29 FSTA has been reloaded and moves to weekly updates
NEWS 24 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update
         frequency
NEWS 25 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 26 Mar 08 Gene Names now available in BIOSIS

NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d,
         CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
         AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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=> s retina-specific nuclear protein
L1          3 RETINA-SPECIFIC NUCLEAR PROTEIN

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          1 DUP REM L1 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 1 ANSWERS - CONTINUE? Y/(N):y

L2  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS
INC.DUPLICATE 1
AN  1991:318007 BIOSIS
DN  BA92:28522
TI  CHARACTERIZATION OF DEVELOPMENTALLY REGULATED AND
***RETINA*** -
    ***SPECIFIC***  ***NUCLEAR***  ***PROTEIN***  BINDING TO A SITE IN
    THE UPSTREAM REGION OF THE RAT OPSIN GENE.
AU  MORABITO M A; YU X; BARNSTABLE C J
CS  DEP. OPHTHALMOL. VISUAL SCI., YALE UNIV. SCH. MED., 330 CEDAR
ST., NEW
    HAVEN, CONN. 06510.
SO  J BIOL CHEM, (1991) 266 (15), 9667-9672.
    CODEN: JBCHA3. ISSN: 0021-9258.
FS  BA; OLD
LA  English
AB  DNase I protection and gel retardation assays have identified a sequence
    5' to the transcription start site of the rat opsin gene that interacts
    with nuclear proteins from mammalian retinas but not from a variety of
    other neural and non-neural tissues. Following sodium dodecyl
    sulfate-polyacrylamide gel electrophoresis and transfer to nitrocellulose
    the protein(s) responsible for this binding were identified with an
    oligonucleotide probe and were found to migrate with an apparent molecular
    size of 40 kilodaltons. The binding complex eluted from fast protein
    liquid chromatography gel filtration as a peak centered at 100
    kilodaltons, suggesting the presence of more than one subunit. Binding
    activity could be detected in postnatal day 1 retinal extracts and
    increased over the next 2 weeks of development, a time course coincident
    with opsin gene expression and maturation of rod photoreceptors. Synthetic
    oligonucleotides with altered sequences showed that the binding was
    dependent upon residues in a CTAAT motif and was facilitated by
    surrounding GGCCCC sequences. The specificity of the binding interaction
    was measured by inhibition of complex formation in a gel retardation
    assay. The unaltered sequence was over 2 orders of magnitude more
    effective at inhibiting complex formation than either an unrelated DNA
    sequence or a concensus sequence corresponding to a known CCAAT box
    binding protein NF1.


=> s retina-specific nuclear receptor
L3          4 RETINA-SPECIFIC NUCLEAR RECEPTOR

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4          2 DUP REM L3 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L4  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2002 ACS
AN  2000:659546 CAPLUS
DN  134:362020
TI  Assignment of the NR2E3 gene to mouse chromosome 9 and to human
chromosome
    15q22.33.fwdarw.q23
AU  Rendtorff, N. D.; Vissing, H.; Tumer, Z.; Silahtaroglu, A.; Tommerup, N.
CS  Department of Medical Genetics, The Panum Institute, Copenhagen, Den.
SO  Cytogenet. Cell Genet. (2000), 89(3-4), 279-280
    CODEN: CGCGBR; ISSN: 0301-0171
PB  S. Karger AG
DT  Journal
LA  English
AB  The human and mouse NR2E3 gene (also known as PNR) encoding a
    ***retina*** - ***specific***  ***nuclear***  ***receptor*** was
    recently identified and found to be a ligand-dependent transcription
    factor. Here we report the mapping of the mouse Nr2e3 gene using
    radiation hybrid mapping, and refine the localization of the human NR2E3
    gene using fluorescence in situ hybridization and radiation hybrid
    mapping. The mouse Nr2e3 gene was mapped to chromosome 9 between
markers
    D9Mit102 and D9Mit207, while the human NR2E3 gene mapped to
chromosome
    15q22.33.fwdarw.q23.
RE.CNT 7    THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
RECORD
        ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4  ANSWER 2 OF 2  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS
INC.DUPLICATE 1
AN  2000:110993 BIOSIS
DN  PREV200000110993
TI  ***Retina*** - ***specific***  ***nuclear***  ***receptor*** :
    A potential regulator of cellular retinaldehyde-binding protein expressed
    in retinal pigment epithelium and Muller glial cells.
AU  Chen, Fang (1); Figueroa, David J.; Marmorstein, Alan D.; Zhang, Qing;
    Petrukhin, Konstantin; Caskey, C. Thomas; Austin, Christopher P.
CS  (1) Department of Bone Biology WP 26A-1000, Merck Research Laboratories,
    West Point, PA, 19486 USA
SO  Proceedings of the National Academy of Sciences of the United States of
    America, (Dec. 21, 1999) Vol. 96, No. 26, pp. 15149-15154.
    ISSN: 0027-8424.
DT  Article
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LA English
SL English
AB In an effort to identify nuclear receptors important in retinal disease, we screened a retina cDNA library for nuclear receptors. Here we describe the identification of a ***retina*** - ***specific*** ***nuclear*** ***receptor*** (RNR) from both human and mouse. Human RNR is a splice variant of the recently published photoreceptor cell-specific nuclear receptor (Kobayashi, M., Takezawa, S., Hara, K., Yu, R. T., Umesono, Y., Agata, K., Taniwaki, M., Yasuda, K. & Umesono, K. (1999) Proc. Natl. Acad. Sci. USA 96, 4814-4819) whereas the mouse RNR is a mouse ortholog. Northern blot and reverse transcription-PCR analyses of human mRNA samples demonstrate that RNR is expressed exclusively in the retina, with transcripts of apprxeq7.5 kb, apprxeq3.0 kb, and apprxeq2.3 kb by Northern blot analysis. In situ hybridization with multiple probes on both primate and mouse eye sections demonstrates that RNR is expressed in the retinal pigment epithelium and in Muller glial cells. By using the Gal4 chimeric receptor/reporter cotransfection system, the ligand binding domain of RNR was found to repress transcriptional activity in the absence of exogenous ligand. Gel mobility shift assays revealed that RNR can interact with the promoter of the cellular retinaldehyde binding protein gene in the presence of retinoic acid receptor (RAR) and/or retinoid X receptor (RXR). These data raise the possibility that RNR acts to regulate the visual cycle through its interaction with cellular retinaldehyde binding protein and therefore may be a target for retinal diseases such as retinitis pigmentosa and age-related macular degeneration.

=> s retina-specific nuclear receptor or RNR
L5 547 RETINA-SPECIFIC NUCLEAR RECEPTOR OR RNR

=> s l5 (10a) (knockout or transgen? or disrupt?)
L6 2 L5 (10A) (KNOCKOUT OR TRANSGEN? OR DISRUPT?)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 1 DUP REM L6 (1 DUPLICATE REMOVED)

=> d bib abs

L7 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2001:526761 BIOSIS
DN PREV200100526761
TI Mutational and structural analyses of the ribonucleotide reductase inhibitor Sml1 define its Rnr1 interaction domain whose inactivation allows suppression of mec1 and rad53 lethality.
AU Zhao, Xiaolan; Georgieva, Bilyana; Chabes, Andrei; Domkin, Vladimir; Ippel, Johannes H.; Schleucher, Jurgen; Wijmenga, Sybren; Thelander, Lars; Rothstein, Rodney (1)
CS (1) Department of Genetics and Development, College of Physicians and Surgeons, Columbia University, 701 West 168th St., New York, NY, 10032: rothstein@cuccfa.ccc.columbia.edu USA
SO Molecular and Cellular Biology, (December, 2000) Vol. 20, No. 23, pp. 9076-9083. print.
ISSN: 0270-7306.
DT Article
LA English
SL English
AB In budding yeast, MEC1 and RAD53 are essential for cell growth. Previously we reported that mec1 or rad53 lethality is suppressed by removal of Sml1, a protein that binds to the large subunit of ribonucleotide reductase (Rnr1) and inhibits RNR activity. To understand further the relationship between this suppression and the Sml1-Rnr1 interaction, we randomly mutagenized the SML1 open reading frame. Seven mutations were identified that did not affect protein expression levels but relieved mec1 and rad53 inviability. Interestingly, all seven mutations abolish the Sml1 interaction with Rnr1, suggesting that this interaction causes the lethality observed in mec1 and rad53 strains. The mutant residues all cluster within the 33 C-terminal amino acids of the 104-amino-acid-long Sml1 protein. Four of these residues reside within an alpha-helical structure that was revealed by nuclear magnetic resonance studies. Moreover, deletions encompassing the N-terminal half of Sml1 do not interfere with its ***RNR*** inhibitory activity. Finally, the seven sml1 mutations also ***disrupt*** the interaction with yeast Rnr3 and human R1, suggesting a conserved binding mechanism between Sml1 and the large subunit of RNR from different species.

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